## Regarding "A prospective study of subclinical myocardial damage in endovascular versus open repair of infrarenal abdominal aortic aneurysms"

We read with interest the recent article by Abraham et al.[1] Perioperative cardiac events are the major cause of perioperative morbidity and mortality in patients undergoing noncardiac vascular surgery due to symptomatic or asymptomatic coronary artery disease (CAD). The incidence of myocardial infarction and cardiac death can be as high as 28% in patients with CAD detected by dobutamine stress echocardiography, whereas myocardial ischemia detected by continuous electrocardiographic recording is more than 39%.[2,3]

In their article, Abraham et al describe a cohort of 149 patients who underwent open (n = 36) or endovascular (n = 113) elective infrarenal abdominal aortic aneurysm repair. Their findings of myocardial ischemia in 25% of patients in the open group are in accordance with these data. For endovascular treatment, there was no information on postoperative troponin T release. Abraham et al concluded that subclinical myocardial damage is probably significantly less in patients treated endovascularly compared with patients who undergo open repair. However, this conclusion can be questioned.

First, as the authors discuss, there is a possibility of selection bias. Apart from differences in vascular anatomy, there are a number of risk factors associated with perioperative cardiac events, defined by the Revised Cardiac Risk Index, and also cardioprotective medication. The Revised Cardiac Risk Index[4] includes several risk factors, ie, type of surgery, history of ischemic heart disease, history of congestive heart failure, history of cerebrovascular disease, preoperative treatment with insulin, and preoperative serum creatinine. The rates of major postoperative cardiac complications ranged from 0.5% to 11% in patients with a score ranging from 0 to 3 or more factors. It would therefore be interesting to know whether there was a difference in the Revised Cardiac Risk Index score between endovascularly treated and open-treated patients. The authors report that there was a difference in the incidence of previous myocardial infarction. However, this is only one of the factors included in the risk index.

Furthermore, medical therapy may influence outcome. Nowadays there is substantial evidence that high-risk vascular surgical patients receiving β-blocker therapy have an improved outcome.[5] Also, recent publications suggest postoperative benefits of statin therapy.[6,7] Because the reported study was a nonrandomized study, patients undergoing endovascular treatment could have been selected on the basis of their supposed increased cardiac risk related to previous events. Therefore, it could well be that patients in the endovascular treatment arm were more frequently receiving cardioprotective medication.

Although the conclusion of the authors is very plausible, for a more accurate comparison of cardiac outcome between endovascular and open treatment of abdominal aortic repair in nonrandomized studies, information on preoperative cardiac conditions, including medication use, objective assessment of CAD, cardiac risk factors, and stratification in risk indices, is of great importance.

*Olaf Schouten, MD*[a]
*Jeroen J. Bax, MD, PhD*[b]
*Hero van Urk, MD, PhD*[a]
*Don Poldermans, MD, PhD*[a,c]

Department of Vascular Surgery
Erasmus MC Rotterdam
The Netherlands[a]

Department of Cardiology
Leiden University Medical Center
The Netherlands[b]

Department of Anesthesiology
Erasmus MC Rotterdam
The Netherlands[c]

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## Reply

We thank Dr Schouten and colleagues for their comments on our article. Their statement in the first paragraph, "For endovascular treatment, there was no information on postoperative troponin T release," is puzzling. The article clearly reports increases in troponin T after endovascular abdominal aortic aneurysm (AAA) repair in 9 (8%) of 109 patients compared with 8 (25%) of 32 patients after open AAA ($P < .02$).[1] Because there was only one patient with clinical myocardial ischemia in the open group and there were none in the endovascular group, our major clinical finding from the study was that there is biochemical evidence of previously underestimated myocardial damage associated with elective AAA repair regardless of the type of repair. This subclinical damage is likely significantly less with endovascular than with open repair. The finding, we believe, is relevant when deciding on the method of AAA repair, even when the patient is considered low risk.

Dr Schouten and colleagues raise the question of differences in risk factors and cardioprotective medications between the endovascular and open groups. We reported that there was no statistically significant difference between the groups with regard to risk factors for ischemic heart disease except for a history of myocardial infarction of 41% in the endovascular group compared with 22% in the open group ($P < .05$). Such a difference should make the endovascular group more likely to develop myocardial ischemia rather than less likely compared with the open group. The Revised Cardiac Risk Index, taking into account cardiac and noncardiac factors, is one of many comorbidity severity scoring systems that may be used. The Ad Hoc Committee for Standardized Reporting Practices in Vascular Surgery[2] states that "At least seven scoring systems have been developed for assessing the relationship of bundled clinical parameters as a measure of cardiac risk." Although it would be of interest to know whether there was a difference in the Revised Cardiac Risk Index between the two groups, it is impractical to analyze the data in multiple ways to fulfill the wishes of all. The recent articles suggesting postoperative benefits of statin

therapy are relevant to the study but, unfortunately, were published after the study period.

We hope that Dr Schouten and colleagues may be able to add to our understanding by using the Revised Cardiac Risk Index and the presence or absence of cardioprotective medication as criteria in a similar study.

*James May, MD, MS, FRACS, FACS*
*Ned Abraham, MB B, MM, FRACS, FACS*

Department of Surgery
University of Sydney
Sydney, Australia

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## Regarding "A prospective study of subclinical myocardial damage in endovascular versus open repair of infrarenal abdominal aortic aneurysms"

We read with interest the article by Abraham et al (J Vasc Surg 2005;41:377-81) reporting increased levels of cardiac troponin (cTn) T in 9% of patients after elective endovascular repair and 25% of patients after elective open repair of infrarenal abdominal aortic aneurysm. These findings are similar to our own, in which increased levels of cTnI were detected in 10 (29%) of 35 patients after elective open aortic reconstruction.[1] Because our study was performed in a unit that did not perform endovascular abdominal aortic aneurysm repair at the time, it seems unlikely that the authors' suggestion that institutional unfamiliarity with open aortic surgery, or more advanced arterial disease in patients unsuitable for endovascular repair, can adequately explain the higher incidence of myocardial injury in the open repair group. One possible explanation for the findings may be related to the fact that there was a higher incidence of previous myocardial infarction in patients treated by endovascular repair (41%) compared with open repair (22%). There is considerable evidence to support the use of antiplatelet agents, β-blockade, and statin therapy in reducing the incidence of early and late myocardial infarction and cardiovascular deaths in patients undergoing major vascular surgery.[2-5] One would expect a cardiologist to have been involved in the management of myocardial infarction in these patients and, therefore, best medical therapy to have been commenced. It is possible that such medical optimization may have contributed to the reduction in myocardial injury associated with endovascular repair. We would be most interested to know whether the authors have information on the relative use of best medical therapy in their two groups of patients.

*Donald J. Adam, MD, FRCSEd*
*Andrew W. Bradbury, BSc, MD, FRCSEd*

University Department of Vascular Surgery
Birmingham Heartlands Hospital
Birmingham, UK

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## Reply

We thank the readers for their comments, and we were very interested to see that they have obtained a similar incidence of cTnI elevation in 29% of patients after elective open aortic reconstruction at a unit that did not perform endovascular abdominal aortic aneurysm repair at the time. This, in our opinion, would confirm our results that there is a statistically and almost certainly clinically significant difference in the incidence of subclinical myocardial damage after the two types of infrarenal abdominal aortic aneurysm repair. In our two groups of patients, the preoperative management did not include routine β-blockade and statin therapy. Although this was not prospectively documented, medical optimization was more likely to have taken place in the open group in view of the degree of severity of the planned procedure. It is quite unlikely that medical optimization would have contributed to the reduction in myocardial injury associated with endovascular repair.

Our study protocol, as approved by the local ethics committee, dictated analysis of all samples in batches in a way that was not related to the day-to-day management of the individual patient. This was to avoid unnecessary interventions based on the results of a serum analysis that under normal circumstances would not take place according to the best current practice of that time, because troponin measurement is not a routine part of postoperative patient care.

*Ned Abraham, MBBS (Hons), MM (Syd), FRACS, FRCS*

Royal Prince Alfred Hospital
Camperdown, NSW, Australia

*James May, MD, MS, FRACS, FACS*

University of Sydney
Department of Surgery
Sydney, Australia

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## Regarding "Ultrasound findings after radiofrequency ablation of the great saphenous vein: Descriptive analysis"

In the recent article by Sergio Salles-Cunha et al,[1] the authors suggest a very high neovascularization rate after radiofrequency ablation (RF) of the great saphenous vein (GSV), which does not correspond to our own experience. The authors describe small vessel networks (SVN), which covers without discrimination all vessels smaller than 2 mm in the surrounding tissue of the treated GSV, including muscular, collateral, and tributary veins and their satellite arteries. The high prevalence of these SVN elements in the groin area and at the thigh level is interpreted as the result of a process similar to the neovascularization described after GSV ligation and stripping. However, without a controlled assessment of